CLINICAL REVIEW

Update on Cardiac Valve Surgery

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INTRODUCTION

Since the clinical introduction of cardiac valvular prosthesis in 1960, valve replacement has saved the lives of hundreds of thousands of patients affected by both rheumatic and non-rheumatic cardiac valve disease. However, when clinicians discovered the consequences of prosthetic valve replacement, it became evident that "valve replacement is simply exchanging one disease for another" (1).

In spite of tremendous strides toward perfecting materials and design, the ideal valve remains elusive. The first valvular prosthesis was developed and implanted in the descending aorta for the treatment of aortic insufficiency by Hufnagel in 1953 (2,3). However, orthotopic prosthetic valve replacement became possible only in 1960 with the advent of the caged-ball prosthesis devised independently by Harken et al. (4,5) and Star and Edwards (6). Newer generations of mechanical prostheses include the monoleaflet Medtronic-Hall (1977) (7) as well as the bi-leaflet St. Jude Medical (1977) (8) and Carbomedics valves (9).

Thromboembolism continues to be a major cause of morbidity and mortality in patients who have undergone mechanical valve replacement surgery. Various follow-up studies have reported the incidence of complications secondary to thrombus formation to be 1% to 4% per patient-year. Such sequelae include embolic strokes or valve malfunction due to impaired disk mobility. Thus, with few exceptions, long-term warfarin therapy sufficient to prolong the prothrombin time to an INR of 2.5 to 3.5 is recommended for patients with mechanical heart valves. The addition of aspirin (100 to 160 mg/day) to warfarin therapy may offer increased protection without increased risk (10).

Tissue valves were developed as a direct response to the thromboembolic complications plaguing the original mechanical valves. The most popular tissue valves, the Hancock and the Carpentier-Edwards prostheses, are porcine xenografts. Thromboembolic rates associated with the use of these tissue valves have been substantially lower than those seen with most mechanical prostheses. In fact, the majority of the patients with tissue valve replacements do not require anticoagulation--the major advantage of tissue valves. The major disadvantage, however, is their lack of durability.

In 1995, Jamieson confirmed previous durability reports by demonstrating that 86% of Carpentier-Edwards valves used for aortic replacement and 70% of those used for mitral replacement were free of structural

deterioration at 10 years. However, these values decreased to 58% and 21% at 15 years, and to 39% and 14% at 17 years for the aortic and mitral prostheses, respectively (11-13). These freedom values are similar to the 18-year freedom from structural valve deterioration (at a younger mean age) reported for the Hancock prosthesis by Bortolotti and associates (14).

From a clinical point of view, the use of bioprosthetic valves in older patients (over 65-70 years of age) is favored because of the increased risk of bleeding in the elderly and the low probability of structural failure of the bioprosthesis in the remaining expected life of these patients. Due to the potential complications associated with implantation of the prosthetic valves, both mechanical and bioprosthetic, the long-term results of valve replacement continue to be suboptimal. Consequently, in the past decade, there has been growing enthusiasm for conservative valve surgery (commissurotomy or valve repair) and for replacement of the pathologic valve with a homograft or pulmonary autograft whenever feasible.

CARDIAC VALVE DISEASE

Mitral Valve Stenosis

Mitral stenosis, in addition to mixed stenosis and incompetence, is usually a consequence of rheumatic heart disease. In the setting of a prominent opening snap and no demonstrable calcification, good results from percutaneous balloon mitral valvuloplasty (PBMV) or open mitral commissurotomy (OMC) can usually be obtained. Under these circumstances, indications for the procedures include NYHA (New York Heart Association) functional class II symptoms and evidence of severe mitral stenosis. The interventions are indicated particularly before the onset of atrial fibrillation (15). Hospital mortality for OMC approaches zero (16,17), but in spite of excellent early and interim results, durability is limited. Valve scarring from the rheumatic process progresses gradually with time, and eventually most patients return with re-stenosis or newly developed incompetence. In Smith's series (14), 88% of patients were alive eight years postoperatively, but 16.1% of them required mitral valve replacement. An additional 8.3% experienced an embolic event. Although OMC appears to result in a longer interval before reoperation, additional follow-up studies are needed to compare the long-term efficacy of PBMV relative to OMC or mitral valve replacement. At present, OMC and PBMV both have a role in the management of selected patients with pliable and noncalcified mitral valves (18).

In the setting of heavy mitral valve calcification, severe subvalvular disease (19), absence of opening snap, previous commisurotomy, or associated mitral incompetence, mitral valve replacement is not indicated in the asymptomatic patient. That is, more severe chronic symptoms (NYHA functional class III) are required to justify surgery in light of the long-term complications of prosthesis.

Mitral Valve Incompetence

Prolapse of a mitral valve leaflet (Barlow's syndrome (20), floppy valve, myxomatous valve degeneration) and ischemic disease are the most common causes of surgically-treatable mitral valve incompetence. Other etiologies include rheumatic disease, infective endocarditis, atrial myxoma, Marfan's syndrome, and trauma.

In patients designated NYHA functional class III
or IV because of mitral incompetence, surgery, whether valve repair or replacement, is clearly indicated. Because of increasing confidence with valvuloplasty, surgery is now generally chosen for patients with mitral regurgitation in NYHA class II and patients showing signs of left ventricular cavity enlargement, even if these patients are virtually asymptomatic. Mitral valvuloplasty yields the best results in valves affected with degenerative disease and the worst results with rheumatic valves, especially in younger patients (21). In comparing mitral valve repair to replacement, Carpentier reported better early and long-term results (15 years) after repair (22). These findings have rendered mitral valve repair, if feasible, the best option for mitral

incompetence at many medical centers in the world.

Since 1991, the author has performed mitral valve repair on 45 patients at the Montreal General Hospital. In this group, there were 28 males and 17 females ranging from 23 to 75 years of age (median: 65 years). Twenty-five patients had myxomatous degeneration of the mitral valve with prolapse of one leaflet, seven had incompetence due to ischemic disease, six had rheumatic disease, five had non-coaptation of the mitral valve leaflet secondary to mitral annular dilatation, three had mitral regurgitation and stenosis, and two had previous endocarditis. Preoperatively, seven patients were designated as NYHA functional class II, 19 as class III, and 19 as class IV.

At surgery, 31 patients underwent quadrangular resection of the prolapsing portion of the posterior leaflet. Seven patients underwent transposition of the posterior leaflet to the anterior leaflet to correct anterior-leaflet prolapse. Three patients had commissurotomy combined with ring annuloplasty, and two had replacement of a chorda tendinea with 5-0 PTFE suturing. One patient underwent translocation of the posteromedial commissure, and one required suturing of a pericardial patch to an anterior-leaflet defect secondary to endocarditis. Every patient underwent mitral ring annuloplasty. A rigid ring (Carpentier-Edwards) was inserted in 36 patients, and a flexible ring (Duran) in nine. Combined with the mitral valve repair, 11 patients underwent coronary artery bypass grafting, three received tricuspid annuloplasty, two had closure of an atrial septal defect, two received aortic valve replacement with a mechanical valve, and two underwent Bentall's procedure. In each case, the cardiopulmonary bypass time ranged from 101 to 148 minutes and the aortic cross-clamp time from 67 to 117 minutes. From 1991 to 1993, the author relied on the epicardial probe, but since 1994 has used transesophageal echocardiography to evaluate valve repair.

Postoperatively, eight patients developed new onset atrial fibrillation, and three required prolonged respiratory ventilation (> 24 hours). Two patients needed exploratory investigation of excessive mediastinal bleeding (one patient bled from an innominate vein tear, and the other bled from a pericardial artery). One patient required a laparotomy one week after valve repair for acute diverticulitis (Hartman's procedure). A 23-year-old patient developed acute pulmonary edema on the fifth day postoperatively. On repeat echocardiogram, he was found to have severe mitral regurgitation and immediately underwent reoperation for mitral valve replacement. However, at the time of the second operation, the author could not explain the failure of the initial repair. Finally, two patients required an intra-aortic balloon pump for left ventricular dysfunction. Unfortunately, these two patients died of cardiogenic shock a few days after surgery.

All of these patients are followed and assessed carefully every six months for two years, and then every year by interview, physical examination, and echocardiogram. Patients in sinus rhythm remain on aspirin for the first three months after surgery. Fourteen patients receive coumadin for chronic atrial fibrillation, and seven take ACE inhibitors. Echocardiogram reveals that 20 patients have no mitral regurgitation, 20 have mild regurgitation, and five have moderate mitral incompetence. None of these patients have a stenotic valve. Two patients experienced systolic anterior motion of the anterior leaflet of the mitral valve that resolved within three to six months post-surgery. Thirty-eight of the patients are now in NYHA class I, and the rest are in class II, with the exception of one patient who died six months after her initial surgery, during a second operation for recurrent severe mitral incompetence. At the time of this second operation in 1991, the author could not explain the malfunction of the mitral valve except to note that the geometry of the mitral annulus was not respected due to the insertion of a flexible ring. Since that event, the author has used the rigid Carpentier-Edwards ring.

Tricuspid Valve Incompetence

Functional tricuspid incompetence secondary to mitral valve disease is the most common cause of tricuspid regurgitation. Other etiologies include endocarditis, traumatic rupture of a leaflet, as well as rheumatic and carcinoid tricuspid valve disease. When tricuspid regurgitation is secondary to mitral valve disease, repair of

the tricuspid incompetence must be considered if the amount of regurgitation is moderate to severe. Because the incompetence is due to dilatation of the tricuspid annulus, most of the regurgitation can be reversed by tricuspid annuloplasty.

Acute tricuspid valve endocarditis is rare and appears to be confined to persons habitually engaging in intravenous drug use (23). Due to the high incidence of recurrent drug use, most replacement devices in the tricuspid position are prone to reinfection. One strategy for avoiding this situation involves performing tricuspid valve excision without replacement (24-27), though this procedure carries a significant risk of hospital mortality (10%) as well as substantial morbidity (23). Fortunately, many of these patients can survive and manage reasonably well for at least 5-10 years, although within this period, at least 20% usually require tricuspid valve replacement to control their symptoms (23). In contrast to the past, whenever tricuspid valve replacement is necessary, mechanical prostheses have been favored, especially for those patients with a good long-term prognosis (28). However, the best surgical treatment of tricuspid valve endocarditis continues to be valve debridement and repair, when feasible. Some of Carpentier's mitral valve repair techniques can be applied to tricuspid valve repair (29).

Aortic Valve Stenosis

In developed countries, adult aortic stenosis most commonly results from calcareous degeneration of congenitally malformed or bicuspid aortic valves. The incidence of rheumatic aortic stenosis is very low.

Any patient with proven aortic stenosis who presents with syncope, heart failure, or angina and is in acceptable general condition should be referred for possible surgery. Balloon valvuloplasty and surgical decalcification have been abandoned because of complications and poor results. The optimal surgical treatment is replacement of the aortic valve with either a prosthetic valve (mechanical or tissue), homograft, or pulmonary autograft. Homograft and pulmonary autograft are usually reserved for children or adults under age 50. In 1967, Ross (30) described the use of an autologous pulmonary valve to replace the aortic valve. The autologous valve was implanted within the aortic root, and the patient's own pulmonary valve was replaced with either an aortic or pulmonary allograft. The viability of pulmonary valve autografts in the aortic root has been documented, and they prove to be more resistant to deterioration than aortic allograft (31,32). Due to the complexity of this operation and the lack of long-term results, Dr. Ross had been the only surgeon carrying out the pulmonary autograft procedure for many years. However, in the last decade, this procedure has been done at numerous centers throughout the world.

In 1995, the author performed the Ross procedure on eight male patients with pure aortic valve stenosis. Their ages ranged from 34 to 62 years (median: 48 years). Perioperative echocardiogram after the procedure revealed trivial to minimal aortic valve insufficiency. One patient (62 years old) developed ventricular fibrillation on the second day post-surgery. Exploration revealed major bleeding from partial dehiscence of the proximal anastomosis of the pulmonary autograft, and although the tear was repaired, the patient died of cerebral hypoxia. The other seven patients have been followed regularly every six months by interview, physical examination, and echocardiogram. All seven are well and medication-free, with competent pulmonary autograft valves.

Aortic Valve Incompetence

Aortic valve ectasia is the most common indication for aortic valve repair or replacement. Other etiologies are rheumatic disease, medionecrosis, congenital malformation, bacterial endocarditis, and trauma. Patients in NYHA class III or IV with clinically significant aortic valve regurgitation are referred for surgery. Similarly, patients who are less symptomatic (class I or II) but demonstrate left ventricular cavity enlargement are also advised to undergo aortic valve surgery. There is a subgroup of patients with aortic valve insufficiency amenable to surgical repair, namely, the patients with leaflet prolapse, perforation, or calcification (33,34).

For other patients, the surgical options are prosthetic valve (tissue or mechanical), homograft, or pulmonary autograft replacement. However, an aortic homograft or pulmonary autograft is contraindicated in any circumstance carrying the risk of progressive dilatation of the aortic sinus or root, since this would produce over-stretching of the homograft or autograft leaflets with resulting central incompetence. Thus, any form of ascending arch aneurysm, aneurysm of the sinus of valsalva, or medionecrotic root ectasia constitutes a contraindication.

FUTURE DIRECTIONS

The Carpentier-Edwards pericardial bioprosthesis represents a new generation of pericardial bioprostheses developed to overcome the complications and failures associated with previous pericardial valve substitutes (35-38). The fully extensible stents and distensible struts decrease shearing stresses on valve leaflets and maintain physiologic aortic ring movements to reduce flow turbulence and vibrations. Infra-stent tissue mounting prevents tissue dehiscence or tears at the attachment site of the tissue to the struts. Glutaraldehyde has been used to preserve the tissue of this new valvular bioprosthesis, which has demonstrated an excellent durability of up to 10 years in the aortic position (39,40). However, more data on long-term durability in the mitral position are needed. Currently, it is the authors' valve substitute of choice when a bioprosthesis is indicated.

The Medtronic company is currently manufacturing two new bioprostheses. One valve is stentless (Freestyle aortic root bioprosthesis) and the other incorporates a stent design (Mosaic bioprosthesis). Both represent new concepts in the manufacture of bioprostheses, specifically, zero-pressure fixation and alpha-amino oleic acid antimineralization treatment. The company Carbomedics is investigating a Photofix bioprosthetic valve: a patented tri-leaflet, central-flow prosthesis, with each leaflet mounted on a flexible support frame. The leaflets are prepared from bovine pericardium treated with a unique patented dye-mediated photo-oxidation process. This new fixation process results in collagen cross-linking without the use of glutaraldehyde, a suspected contributor to the calcification failures of other clinically available tissue valves. A human implant took place in December 1994 in Germany, and this patient is currently well. Over time, clinical experience with the Photofix, Freestyle, and Mosaic valves will accumulate.

In conclusion, the patients with new-generation mechanical valves, anticoagulated to maintain an INR of 2.5 to 3.5, experience less thromboembolic and hemorrhagic complications than reported with older mechanical valves. The development of an ideal mechanical valve that would require minimal anticoagulation or antiplatelet therapy alone still awaits. In the meantime, the use of pulmonary autograft or homograft in the aortic position is a good substitute. Based on limited experience (41), it can be suspected that, in the near future, replacement of the mitral valve by a homograft will prove advantageous.

The authors certify that they have no affiliation with or proprietary interest, financial or otherwise, in any organization, manufacturer, instrument, or procedure discussed in this article.

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